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## Living History-Biography: A Rambling Rationalist

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### PROFESSIONAL SUMMARY

**Born March 30, 1917, Rio de Janeiro, Brazil**

Graduated in Natural History (1938) and Medicine (1941), PhD in Biology (1953), Staff member (1958), Privat Docent (1963), Associate Professor (1973), and Full Professor (1978) at the University of São Paulo.

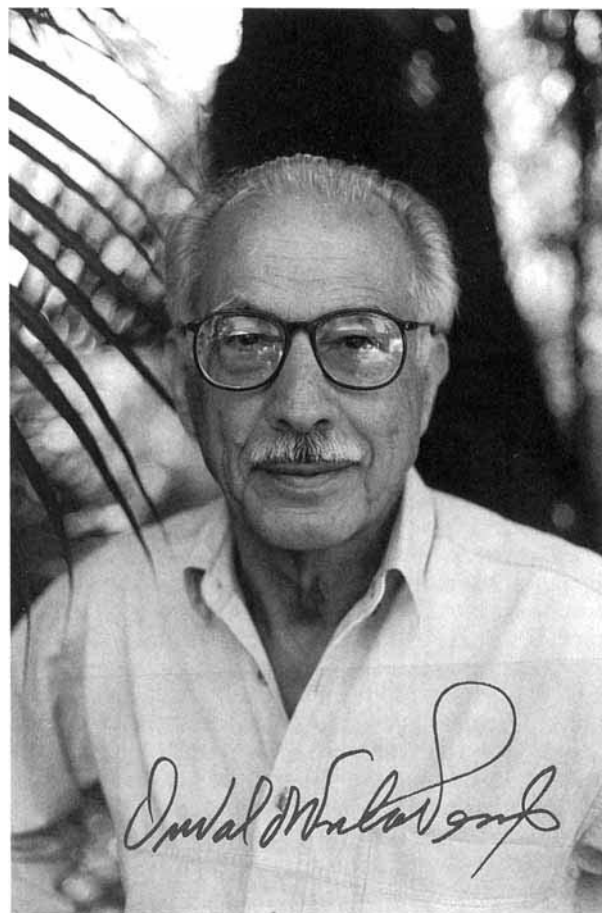
Member of the Academy of Sciences of São Paulo State (1974) and of the Brazilian Academy of Sciences (1979); member ("Comendador") of the Brazilian National Order of Scientific Merit (1995); Professor Emeritus of the University of São Paulo (1995).

Fellow of the Rockefeller Foundation at Columbia University (1953–1955), and of the Department of State (Fullbright), as Visiting Professor at the University of Wisconsin (1964–1965).

Secondary teacher of Sciences and Biology in public schools in Rio de Janeiro (1939–1958), staff member at the University of Brasil (Rio de Janeiro, 1942–1958).

Expert in science education of the Pan American Union, Washington, D. C. (1955–1956); consultant on human genetics of the World Health Organization (1961–1986); director of the Brazilian Center of the Multinational Genetics Program of the Pan American Union (1968–1973); director of the Biology Curriculum Study at the University of São Paulo (1972–1979); president of the Brazilian Society of Genetics (1978–1970) and the Latin-American Genetics Association (1969–1971).

Prizes: Brazilian José Reis Prize for Popularization of Science (1981), UNESCO International Kalinga Prize for Popularization of Science (1982), Alfred Jurzikowyski Prize of the Brazilian National Academy of Medicine, for relevant basic research for medicine (1989).



My father published a book of sonnets when he was 22 and my oldest brother was a poet. Since I believe that the management of words is a multifactorial trait, I consider these facts related to my being the only geneticist ever to succeed in publishing a sonnet in the

*American Journal of Medical Genetics* (A88). Another peculiarity pervasive in my father's family is the need of taking any train of thought (right or wrong) to a coherent end and accepting the consequences. As an adolescent, my father was all the time pushing his only brother to practice religion in earnest. A few years later my father became an atheist and his brother a priest. I lived an abbreviated recapitulation of this history. Not having been baptized at birth, I demanded this privilege at 10, spent a few years in exalted mysticism and

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became an atheist for life. I am assertive and sure of my ideas and, if I change them, this does not make me less positive. I am afraid I am, more than anything, a hard-nosed rationalist.

I have other defects that helped me a lot. I have always had a bad memory for names and isolated facts. This obliges me to rely on relationships and analytical thinking. Another thing: since everybody perceives at once that I am a bad administrator, I have been spared the loss of much time with bureaucracy.

### THREE PATHWAYS

I was born in 1917, in Rio de Janeiro, at that time the capital of Brazil. The Universidade do Distrito Federal was founded there in 1935, just in time for me and 17 other youths to join its first class of Natural History (biology plus geological sciences). We enjoyed the best course ever given in Brazil for a simple reason. The organizer of the University, Anísio Teixeira, an outstanding educator, nominated to each chair the best researcher in the country, even if he had never delivered a lecture. When the Brazilian best was not good enough, he would send for a European scholar. The result was that most of our professors had a straightforward notion about teaching: since research was the thing they knew how to do best, they put us doing it from the first day of class. We made field trips, collected and determined specimens, consulted publications, even if it amounted to a struggle, as in my case, with Latin original descriptions in Martius' *Flora Brasiliensis*.

Our course, largely centered on the activities of the students, gave me a training in taxonomy that allowed me to publish several papers on Brazilian *drosophilidae* (A3-7, 9-10) even before my PhD thesis (A12). In addition, it made me a fanatical proponent and user of better teaching methods, launching me on a life-long career as a specialist in science education.

The third branch of my tripartite road started when I published in a popular magazine, in 1938, a free lance article called *Por que os filhos se parecem com os pais?* (Why do children resemble their parents?). My ability to write about science for the general public and for students and teachers improved through practice and over the years I became the author of more than 700 articles in newspapers and popular magazines and many textbooks (D1-30).

### TEACHING HOW TO TEACH

I taught part time science in high schools for 20 years and gave summer courses for science teachers almost all my life since 1939. I also wrote extensively on science education (D3, 5, 20-22, teacher's guides for D8-19, and articles E1-42).

In 1956-1957, I interrupted my academic career to work full time in the Pan American Union (OAS) in Washington, DC, as an expert in science education. When I was hired, I presented to my director a plan of action, which he approved enthusiastically: it consisted in organizing courses to form leaders who would repeat the courses to form more leaders in a geometric progression. In this way I hoped to improve perceptibly science teaching in some Latin American country.

I would visit my director periodically to find out about the funds needed for starting my program and he would ask for a little more time. As the weeks passed, I started wondering if perhaps I should return to the University, since I was losing my time. Then I had an inspiration and asked my boss if I was allowed to use my spare office time to work on a textbook. Since he felt uncomfortable with his failure in getting my program started, he agreed wholeheartedly and was bothered no more by me. My program never got started, but when I left OAS my *Biologia na Escola Secundária* (D2) was almost ready. It was published in 1960 by the Ministry of Education and had also a number of commercial editions.

The *Biologia* . . . had a role in Brazilian education comparable, on a smaller scale, to the one exerted by the books of the Biological Sciences Curriculum Study (BSCS) in the United States a few years later. It was widely used in senior high and in the preparation for college entrance examination through the sixties and seventies. Many people have told me how the book influenced them toward scientific careers. Lately, these people tend to have paunches and whitish hair, or they tell me: "You know, my father studied your book; we have it at home to this day."

This success was mainly due to the problem approach. Starting a subject, I often presented an event or situation of interest to the youth, raised a problem, and, in the process of discussing it, I would bring in facts and principles. In the first unit, I sketched better methods of teaching and studying, so that teachers and students alike could keep a critical eye on the performances of all. The virtues of discussions instead of lectures were stressed. Later I expanded this unit to be an independent book on teaching biology (D5), published in Spanish by OAS and followed by another one, in Portuguese, on teaching sciences (D3). Now I feel that, with these books, I contributed to improve science teaching in Latin America more than I would have, had my OAS program of tandem courses been developed.

In 1961, my colleague Myriam Krasilshik and I spent one month in Boulder, as foreign collaborators in the last writing conference of the BSCS, under the coordination of the geneticist and educator Bentley Glass (with whom I had collected data on hyperextensibility of the thumbs, of which he was an extreme example). Back in Brazil, Krasilshik and I published an adaptation of the ecological part of the BSCS Green Version, substituting Brazilian species for the American ones in corresponding eco-dramas (C4-5).

I also produced, with collaborators, a version of the *Biologia* . . . for use in the Northeast of Brazil (D7), fitting in it subjects of special interest to the populations in that arid region, and five courses, accompanied by teacher's guides, on different sectors of biology (basic, agronomical, veterinary, medical, and laboratorial), meant to help students to discover their specific inclinations, if they liked biology (D23-27).

I was on the Organizing Committee of the First Interamerican Conference on the Teaching of Biology (San José, Costa Rica, 1963) (E12, 15) and of the Second one (Asunción, Paraguay, 1972) (E22, 23), which was the last to this day. The decade between them was very

active with respect to the movement for improving science teaching in Latin America. Workshops on different aspects of teaching biology and Conferences on the teaching of the other basic sciences were also sponsored by the OAS. I was involved in some of them.

As a member of the Expert Committee on Human Genetics of the World Health Organization, I was a co-writer of a report on the teaching of genetics (E10). Representing OAS, I participated in the Seminar on the Reform of Biology Teaching, sponsored by the European Organization for the Economic Cooperation and Development (Vevey, Switzerland, 1962), and was a co-writer of a technical workshop on the teaching of biology (Montevideo, Uruguay, 1971) (E19). In 1966, as a member of a OAS team, I visited five South American countries surveying the status of science teaching in them.

Most of my textbooks have been written with collaborators and served as a training for new writers: 21 junior authors have worked with me. I also edited (section C) five books with one to three coeditors and translated into Portuguese six books, including L.C. Dunn and Th. Dobzhansky's *Herança, Raça e Sociedade* (C1), V. McKusick's *Genética humana* (C9) and, J.M. Opitz's *Tópicos recentes de genética clínica* (C12), translated from his unpublished lectures in English, presented in São Paulo.

### WHY SCIENCE?

My family was oriented toward pedagogy and literature, with a taste for mathematics. My mother was a primary teacher. My father, after his start as a poet, was a lawyer and pamphletary politician for a while and finally settled as an education administrator and journalist. He wrote three books on education and for 14 years maintained a daily column on education in the newspaper *Jornal do Brasil* in Rio. In his youth, he had attended the engineering school (before shifting to law) and, in 1906, published an article on systems of numeration other than the decimal. My two brothers were also in engineering school, but did not complete their courses, the first because he became ill and the second because of his decision to work instead. My only sister (I was the last) dedicated herself to elementary education.

My inclination toward science was awakened in high school by a teacher who was an inspired naturalist along with being a professional pharmacist. He used to take us on field trips and show us how to observe, collect, and classify specimens. His discussions in class were based on first hand knowledge in various fields. Because of him, I decided, with four other colleagues in my class, to study medicine, as a way toward dealing with biological subjects, since, at that time, no courses existed in Brazil for preparing specialists in basic sciences.

When we were finishing the first year in medical school, a fortuitous encounter downtown with one of my colleagues changed my future professional life. He said: "Gee! I just learned that a course for training naturalists is being opened in a new University here in Rio. They accept candidates only until tomorrow. Let us join it?" We summoned three other pals and all of us signed up for it. The aim of the course was to make good science teachers and, hopefully, research naturalists.

We immediately fell in love with the course. Since the new University had as yet no buildings, we would meet in our teachers' labs or in classrooms borrowed from the medical and engineering schools.

Most of our teachers used the project method. Our botany professor, Alberto Sampaio, was the head of the Plant Division of the National Museum of Natural History, installed in the huge palace of Emperor Dom Pedro II, made vacant by the proclamation of the republic. Sampaio had collected all over Brazil but rarely had given a class. Accordingly, he received us with a list of plant families selected for having few genera in the State of Rio de Janeiro, claiming that they were not well studied and exhorting us to divide into groups and make monographs on them. This accounts for my first scientific paper, published before graduation (A1).

Our zoology professor, Lauro Travassos, from the Instituto Oswaldo Cruz, was the best taxonomist in the country. At our first meeting, he gave us the assignment to collect insects from ten different orders, dissect their bucal apparati and draw the innervations of a variety of wings. Having explained this, he bid us farewell adding that, if we worked diligently, we would be prepared in a month's time for his first class in entomology.

He realized the importance of genetics and, since it was not included in the curriculum, asked a colleague, who was well read on it, to give us a few classes on the subject. The lecturer gave us copies of Mendel's paper to read, comment and discuss. I became so interested in the origins of genetics that my second published paper was a review of the notions of inheritance in ancient Greece and Rome (B1), followed by a mathematical analysis of Mendelian segregation (A2) and a genetic interpretation of the law of recapitulation (A8). These, together with my first popularization article, could be taken as the prenatal signs of a geneticist. However, before blossoming as such, I had yet to go through years of taxonomy.

### FIRST JOBS

My father liked chess. We keep to this day the complete collection of the French magazine *La Stratégie*, from 1867 to 1926, which he owned. I remember following games from it at age 7 years when it was a matter of staying in bed with a cold. At 19, at first with the help of my father, I started a weekly chess column in the newspaper *Jornal do Brasil* and continued it for 5 years. The high level of the column was mainly derived from *La Stratégie*. Although I practically never played chess after this period, now, in my old age, I am appreciating it again as a gratifying hobby.

Eventually I succeeded in changing the subject of my column to science and maintained it for 7 more years, under the heading of "A Ciência em Marcha," while also writing occasionally for other newspapers.

In those early days (1949–1952), I revised the science part of the *Youth Treasure*, the Jackson's Cyclopeadia (12 volumes), for a new edition in Portuguese and wrote a volume on Biología for the *Enciclopedia Práctica Jackson*, published in Spanish (D1). More recently (1975) I was in charge of planning and reviewing 230 entries about biological matters in the 20 volumes of

the Brazilian *Enciclopédia Mirador Internacional*, in collaboration with Lidia Rosenberg Aratangy. All this gave me a good understanding of the ways of science and improved my skills writing about it.

My first job as a biologist arrived, by indication of my zoology professor, when I was in the last year of my Natural History course. It was in a fishery being installed by the Ministry of Agriculture in Pirassununga, São Paulo. During the construction of the buildings, we worked 6 months part time in Rio studying ichthyology and organizing a catalogue of Brazilian fresh water fishes. The next 2 months were spent in Pirassununga, studying the Mogiguassu River inhabitants and trying to induce some of them to reproduce in captivity by injecting them with macerated pituitary glands extracted from some of their pals.

Our chief, Rodolpho von Ihering, was a inspired zoologist who had introduced fishes to the artificial lakes constructed in the Northeast of Brazil to counteract the periodic droughts typical of that region. He was a compulsive worker who, having a good idea, would not pause a moment before putting it into action. He despised red tape to the point of hiring nine of us by word of mouth, based solely on a vague agreement from the Minister of Agriculture, who happened to be his friend. As a result, we worked many months without seeing the color of money, taking it in our stride, since we lived with our parents. However, a senior zoologist, who had immigrated with his wife from Germany and had also been "hired," displayed recurrent fits of anger over our predicament, which amused us. Finally an expert on budget, summoned by the Minister, found a way of backpaying us "legally" in cold cash against our signature on a document that we found prudent not to read. R. von Ihering was a top science writer for the layman. He made us contribute articles about fish biology to a magazine read by farmers and showed us how to do applied research even under unfavorable circumstances.

In the middle of 1938, immediately after my graduation in Natural History, I was nominated to teach in the public school system of Rio. Since this was part time, I kept my job in the first phase of the fishery project. Its second phase coincided with summer vacations, but after that, I had to give it up to stay in Rio.

I taught biology in a female professional school, which offered crafts, such as sewing, embroidery, cooking, and female hat manufacture, but included a full academic curriculum. Arts were represented by a choral and a ballet group. The inescapable result of this scenario was that in December 30, 1930, at 22 years, I was a married man.

Besides teaching, I spent the 2 following years mainly attending a course on biological sciences applied to medicine, given at the Instituto Oswaldo Cruz, our leading tropical diseases research center. This was more exciting and inspiring than medical school, which I continued anyway to a happy and incompetent end, in 1941.

In 1943, a few colleagues and I tried to conciliate our dichotomic training in biology and medicine by starting a laboratory for clinical analyses. Two factors terminated this apparently logical move very quickly: our lack of contacts within the medical profession and our

ignorance of the subject. Fortunately, the scarcity of patients and the simplicity of the tests requested prevented this adventure from resulting in disagreeable consequences.

### DOBZHANSKY APPEARS

One day I heard that a great scholar was coming to Brazil to study fruit flies. We knew, of course, the importance of drosophila for genetics, but this was about all. An entomologist I asked about them said: "Look, these are North American flies; they don't exist here." Notwithstanding, in 1943 I found myself in André Dreyfus' laboratory, at the University of São Paulo, dealing not only with living native flies, but also with Theodosius Dobzhansky himself. He was working with Crodovaldo Pavan on a classification key to determine Brazilian drosophilae, among which they were describing many new species. They wanted to check the key using naive entomologists and Travassos, being a friend of Dreyfus, indicated me and a colleague for the task. We spent 2 weeks at it, maintained by a fellowship.

The impression Dobzhansky gave me was exhilarating. He displayed a happy exuberance. Research was his greatest enjoyment and his entire personality was tuned to it. He would make a point of being one of the crowd even among young disciples, but his natural authority was always clear and assuring. He was assertive, but not pompous. He worked intently, conceding himself brief moments of relaxation only because he believed that they would enhance his performance.

As a taxonomist, I had jumped from saxifragaceae to fresh water fish and now I was tasting drosophilae. Then. . . .

### PT

Having already had a daughter (Sonia, born in 1942, who is now a professor of physics), my life was brutally disrupted: I had a hemoptysis and was diagnosed as an advanced case of bilateral pulmonary tuberculosis.

My oldest brother had died of this disease after wandering 7 years through sanatoriums and places of "good climate." A number of my cousins on my mother's side had also been affected, most of them dying from it. No cases occurred on my father's side of the family (which, on the other hand, had its share of cases of mood disorder). A few years ago, my sister, now 83, was treated and cured of a tuberculous nodule in her lung, taken at first as cancerous. My family inheritance was working on me.

Since drug treatment was not available at the time and my doctor considered pneumothorax too risky for severe bilateral cases, he advised me to go to Campos do Jordão, a mountain resort in São Paulo state, and stay there resting as long as I could (probably meaning forever).

If I am writing about this now, it is because I met there a recently arrived young doctor, Mozart Tavares Lima, who was studying the subject thoroughly (with the primary intent to cure himself). He put me immediately through an unilateral pneumothorax, followed by Jacobeus surgery to eliminate adhesions and, after a few months, repeated the whole process on the other

lung. In due time I improved and went back to Rio to resume my activities, with the added obligation to go twice a month for 3 years to my old doctor to replenish with air alternatively the two pneumothoraxes. With this, I was finally cured.

### THE FLIES

Having to stop my activities in order to rest in Campos do Jordão, I searched for something to do while doing nothing and found it. I hired a street urchin to put a trap can in the bushes near my house and bring it to me later, covered with a net. In this way I got plenty of *Drosophila* specimens without moving from my chair. The Museu Nacional, where I had been a voluntary researcher, lent me an old entomological microscope and Duda's treatise on the South American drosophilidae. I was set to work in what would become my PhD thesis: a revision of the tripunctata group of *Drosophila* with descriptions of fifteen new species, defended in 1954 (A12).

Once back in Rio, I resumed my teaching and, with students, started a research group in the Biology Department headed by A.G. Lagden Cavalcanti of my alma mater, the Universidade do Distrito Federal, transmuted in the Universidade do Brasil. Hans Burla, an excellent Swiss drosophilist who had spent the year of 1944 in São Paulo as a member of Dobzhansky's group, joined us for another year. In 1947 I became a part time member of the Department of Biology.

### COLUMBIA UNIVERSITY

In 1960, the First South American Symposium on Genetics was held in São Paulo in honor of Dr. Harry M. Miller, Jr., associate director for biological sciences of the Rockefeller Foundation, who, with immense dedication, had been the main supporter of the development of research in Latin America. I was one of the many beginners launched with a fellowship from him, which, in 1953, brought me to Dobzhansky in New York.

By that time, I was separated from my first wife, Elisa, a physicist, with whom I had had also a second child, Roberto, now a surgeon in Rio.

Once Dobzhansky had told me during a field trip in Rio: "My dear Frota," he said, "in order to study Brazilian drosophilae properly, you must come to New York!" The prophecy was confirmed: this was my first opportunity to do full time research. The nature of my activity had also changed. I was crossing flies and looking for results, instead of describing and classifying them.

Besides, I was in an active cosmopolitan laboratory, enjoying seminars on new fields (like those by F.J. Kallmann on psychiatric genetics), meeting important scientists, and chatting with graduate students and distinguished Dobzhansky's disciples such as Bruce Wallace. On the other side of my bench worked Richard Lewontin, always full of wit, and later Phillip Shepard, who made me develop an appreciation for British humor. It was a delightful and inspiring environment.

Dobzhansky was the perfect boss. He rejected all kinds of reverences and gave us the right amount of freedom. Since he spent several hours a day at the microscope determining frequencies of translocations in different populations of *Drosophila pseudoobscura*, an

automatic and boring activity, he frequently called one of us to sit by him and chat while he worked. He did not like his obvious domineering personality and tried hard to suppress its manifestations.

My project consisted in crossing various strains of *D. pseudoobscura* to demonstrate heterosis, an aim which I failed to reach repeatedly. This was especially embarrassing because another researcher in the group was obtaining the expected results. We matched our strains, compared our techniques, and ran a twin experiment, to no avail: he got the desired results and I did not. To this day I do not know who or what was wrong.

Fortunately, during my intimacy with *D. pseudoobscura*, I noticed some flies exhibiting discolored testes instead of red ones and was able to demonstrate that this was the effect of an X-linked mutant gene (A13), the first to be known in *Drosophila* which does not affect at the same time the color of the eyes. It is appropriate to consider this as my baptism as a geneticist. It was also my last article on *Drosophila*.

I also carried out a little project (not published) to select for the number of abdominal bristles in *D. ananassae* and arrived at a difference of about 20 bristles between the plus and the minus lines. This gave me direct insight on the flexibility of polygenic systems.

### THE GRAND TOUR

Crowning my training with Dobzhansky, I was sent to visit about 20 laboratories (in 1 month!) around the US, to become acquainted with the leaders of genetics research.

In Bloomington, J. H. Muller invited me to give a seminar about the work I had done in New York. I was enchanted with him and 10 years later I was delighted to meet him again at the University of Wisconsin. We were together again at a congress on Science Education in Switzerland. There he was, the most illustrious participant, talking very assertively on how to teach, only to be rebuked by an English educator in such terms as: "Dr. Muller, if we do as you preach, most irreparable damage will be done to the minds of our students!"

In Austin, I was received regally by M. R. Wheeler, in the capacity of a dear colleague, since I had published with him a revision of the drosophilid genus *Neotanygastrella* (A10).

I visited J. Clausen, in Palo Alto, and he invited me to his house. After dinner, his wife showed me a table cloth embroidered with the signatures of half a hundred biologists who had visited them and asked me to put my name on an empty spot, so that she could trace it over with needle work. I wonder if this original piece of recollection has survived.

In Chicago, I walked along a quiet corridor at the University looking for Sewall Wright's room for an unannounced visit. His door was ajar and I could see him alone, working very intently at his desk. On an impulse, I went away, failing the only purpose of my trip to Chicago, rather than perturbing this sacred scene. I was compensated later for my discretion by being able to listen to his conversations at lunch time gatherings during my stay at the University of Wisconsin, where he worked after retirement.

## TURNING TOWARD HUMANS

My interest in human genetics arose when a student asked me why there were so many deaf people in Goiás. He knew this for a fact because he worked part time in the Brazilian Census Bureau, which recorded deafness and blindness at that time. My answer was "let us go there and find out." We went to the epicenter of the concentration of deafness, Planaltina county, not far from where Brasília was constructed later on. We surveyed about two hundred families constituting the main village in the county, asking about consanguinity, birth defects, and other diseases. We found 19% consanguineous couples, 7% being first cousins (A15). We discovered that we were in a goiter region and arrived at the tentative conclusion that consanguinity, although high, could not be the sole explanation for the prevalence of deaf-mutism in the region, which probably resulted chiefly from goiter in the mothers.

On our way back, we stopped in Goiânia, the capital of Goiás, and met there an avid genealogist who allowed us to determine consanguinity rates in his enormous pedigrees of traditional families. He was kind enough to leave his registers with us overnight, since we were leaving town next morning.

Back in Rio, I was summoned by the police under the accusation of having stolen the genealogist's records. He worked in the police department in Goiânia and had asked his colleague in Rio to arrest me. The confusion arose because I left the material at the hotel, as agreed, but the clerk forgot to notify his substitute, who told the genealogist, when he appeared for the papers, that nothing had been left for him. A few cables clarified the situation, but I learned that dealing with humans involves more risks than studying fruit flies.

I never attended a formal course on genetics besides the small introductory study of Mendel's paper I mentioned. This made each project I attacked an adventure in the unknown, along which I had to look for information in books and articles. Giving classes on genetics was also an effective way of learning the subject, since, as a colleague used to say with no great exaggeration, in a course, the only person that really learns is the teacher.

In the forties, I was already teaching genetics part time at the Universidade do Brasil and I had an undergraduate student, P. H. Saldanha, who did not like working with *Drosophila* and carried out instead, as a course project, a survey of the human sensitivity to phenylthiourea. He learned about the publication of G. Dahlberg's book, *Mathematical Methods for Population Genetics* (1948), and wrote asking for a free copy of it. It was sent and Saldanha and I enjoyed studying it. After graduation, he continued research along the same line and on blood groups and published three papers, in 1956, as the beginning of a successful career as a human geneticist.

Before that, in 1952, N. Freire-Maia, the pioneer of Brazilian human and medical genetics, published two papers on consanguineous marriages, inaugurating modern Brazilian human genetics. He had explained to me the concept of "size of isolates," by writing formulae

on the sand, since our meeting was taking place at Flamengo beach, in Rio. This provided the inspiration for my subsequent work on isolates. Most human geneticists in Brazil had been trained first as *Drosophila* philists, under the influence of Dobzhansky. This explains why our tendency in the beginning has been mainly population.

In 1957 I gave my first human genetics seminar talking about my investigation on deaf-mutism and goiter in the state of Goiás. This was at Ann Arbor, where N. Freire-Maia and F.M. Salzano were working with J.V. Neel. Thus three Brazilian human geneticists were in the conference room, more than could be found that day in the whole of Brazil.

## CENSUS DATA

Our survey on deafness in Goiás called my attention to the possibility of extracting useful data for human genetics research from National Censuses. When working with the OAS, in Washington, I could not continue laboratory work and looked for some theoretical project for my spare time. It occurred to me that perhaps I could estimate the "size of isolates" from the average number of cousins per person for each region, calculated from census data, combined with consanguinity rates.

I made an amendment in Dahlberg's formula and worked out a way for obtaining the average number of cousins from census data. For consanguinity rates, I resorted to the estimates, for each Brazilian state, made by Freire-Maia from Catholic Church records. This idea led to my second paper in an American journal (A14), and to my Privat-Dozent thesis (1963), where the influence of geographical and social factors on the degree of isolation of Brazilian populations was analyzed (A61).

Also by statistical manipulations of Census data, we were able to estimate prevalences at birth of Down syndrome for each state of Brazil and for rural and urban zones, in collaboration with Nilda Martello, whose Master thesis on consanguinity, in 1967, was the first I directed. We showed (A59, 60) that direct records of prevalence of Down syndrome in maternity hospitals in Brazil are biased not only because of uncertain diagnosis at birth but also because aged mothers are under-represented in maternity samples. Indeed, being experienced, they tend to deliver at home more than younger mothers. We extracted from census data the average number of children delivered by mothers of each age and applied to it the literature risks for Down syndrome determined directly for children of mothers of each age. In this way we could estimate, for each Brazilian state, the frequencies at birth of Down syndrome children, separately for rural and urban populations (A37, 99, 103). We also analyzed the influence of different social, economical, and cultural factors in the variation of the prevalences, at birth, of Down syndrome cases as well as their historical trends.

## DAMAGE FROM CONSANGUINITY

Consanguinity attracted the attention of our first human geneticists because it used to be high in Brazil in



contrast with the present breaking of the isolates that our populations are experiencing. The subject was studied thoroughly by N. Freire-Maia and his collaborators by means of direct surveys in varied types of populations and by resorting to Catholic Church records. With the first method they assessed the effect of consanguinity on the frequency of birth defects and diseases and also studied the impact of racial and social variables on it. The second method allowed them to sketch a consanguinity map of Brazil and describe its historical trends.

I coordinated a project developed by 23 human geneticists aimed at confirming Freire-Maia's consanguinity map by a different method. We surveyed 1,000 college students in each of 22 universities (one in each state capital). The students responded whether their parents had some known consanguinity. Those answering yes would sketch his or her pedigree helped by the geneticist, setting clearly the degree of relationship. The results gave general agreement with those of Freire-Maia, based on Church records, with small discrepancies attributable to methodological weak points which differ in the two methods.

Using our estimates of consanguinity for each state capital and Freire-Maia's for Brazil as a whole, and obtaining the annual number of children born alive from the Census, I was able to estimate (A113) the damage resulting from consanguinity in the corresponding populations, as measured by the number of children born per year who are abnormal due to parental consanguinity. This was compared to the damage, measured by the annual number of children being born with Down syndrome to women 35 or more years old (census method). The values, estimated for the seventies, are about 3600 for consanguinity and 5500 for the maternal age effect. Fortunately both types of damage are dropping very fast due to decreased consanguinity and modern family planning.

### MOVE TO SÃO PAULO

When I was working at the OAS, C. Pavan, the head of the Department of Biology at the University of São Paulo, visited me in Washington (1956) and invited me to join his staff. I was already convinced that my position at the OAS would never transcend a bureaucratic activity for lack of funds, and was inclined to leave. My second wife, Elizabeth, an American physicist, worked at the Bureau of Standards, but agreed to our return to Brazil. In Rio, I resumed my work at the University and also at a school training primary teachers.

One year later (1958) we moved to São Paulo and I started to work in the same building where I had met Dobzhansky for the first time 15 years before. It was a completely new life, inaugurated with the birth of our first daughter Vanessa, followed, the next year, by that of Osvaldo Jr. (who has obtained a PhD in philosophy of science in Bloomington and now works in São Paulo).

In spite of its name, the Department of Biology concentrated only on *Drosophila* work, but I had the freedom (and the difficulty) of deciding what to do: *Drosophila* or human genetics. I had ambivalent feelings trying to make a decision until I remembered a dic-

tum which ran in my family, to the effect that dilemmas find their best solution with time. With this, I forgot the issue and, in due time, I found myself involved exclusively with human genetics.

My colleagues in São Paulo received me with great friendship and warmth and C. Pavan has been, since the beginning, a dedicated promoter of the new field in his department.

Saldanha was already in São Paulo, where he founded the Chair of Medical Genetics in the School of Medicine, the first to come into existence in Brazil. I could count, therefore, on a colleague and friend to help attack the new subject.

Reasoning that Brazil lacked human geneticists and it was part of our task to form them, we organized a full-time 3 month course for biologists or physicians from different states of Brazil plus a Chilean doctor and summoned whoever was available to give classes, including N. Freire-Maia, F.M. Salzano, Phyllis Eveleth, an American anthropologist residing in São Paulo, and the hematologists F. Ottensooser and P.C. Junqueira. The course ended with a week of field research in a Dutch rural racial isolate (A16) and formed a number of human geneticists who became leaders in the field, such as B. Beigelman, A. Freire-Maia, H. Carvalho, and N. Leon.

My association with Saldanha (A17, 19) led me to a line of theoretical research mainly on mutation rates (A18, 22, 23, 26, 40). Analyzing original data collected by hematologists in Rio, I also demonstrated dosage compensation in the case of hemophilia B, in agreement to Lyon hypothesis, my only paper published in *Science* (A20).

### THE INTERNATIONAL SET

One day I received a telephone call from Rio summoning me to meet there a representative of the WHO, Dr. R.N. Dobson, who was visiting Brazil. When I got there, I learned that he had not returned yet from the beach in Copacabana, where he was bathing with his wife and children. Being a good "carioca," I never go to Rio without taking with me bathing trunks, so I decided to meet him at the beach. We talked about genetics for hours in a relaxed mood, taking now and then a dip in the water, wife and kids cavorting around. No doubt this contributed to the invitation I got a little later to join the WHO Expert Committee on human genetics, to which I belonged from 1961 to 1986, being appointed every 5 years.

I had already participated in a seminar on the use of vital statistics for genetics and radiation studies and in sessions of the Scientific Committee on the effects of atomic radiation, both promoted by UNO, but now I was called to integrating writing groups, which produced publications: one was on the teaching of genetics (E10), others on applied genetics (A25, 44). During the meetings, I enjoyed the company of outstanding geneticists from different countries, appreciated their culture and style, and participated in their gossip.

I learned first hand of the inactivation of the X chromosome from the mouth of J.A. Fraser Roberts who, arriving at the WHO where we waited for him, started

shouting from a distance in great excitement: "Do you know what Mary Lyon told me, in the London airport? She just proved that . . ."

I felt proud, in one of the meetings, because my conciliatory stance restored peace when a brawl started because the WHO officer in charge of accompanying our work declared that our report would simply not be published if we insisted on mentioning the importance of family planning. This was because the General Assembly of the member State, following the stand of the Catholic Church, had voted prohibiting WHO to even mention the subject. One of our colleagues became indignant with the interference by the officer and threatened to abandon the committee immediately. I succeeded in sidetracking the issue by observing that our recommendation, although appropriate, was not directly connected with the subject we were elaborating, genetics and public health, and therefore could be set aside. It was gratifying to see WHO change its position later on, becoming a champion of family planning.

Years later I adopted an attitude similar to that of our colleague when I was one of the editors of a book on genetics of human populations that UNESCO wanted to publish in three versions, for Latin America, Africa, and Asia. We were surprised by an order to suppress from the chapter on human variation any explanation about race or even the use of this word. It came from a censorship committee in charge of assuring that nothing was published under UNESCO's name against the determinations of its General Assembly, which had ruled that the subject was forbidden. Since I always had been interested in the concept of race and its biological, evolutionary, and social implications (A11) as I am now (A128, 129, 130), I could not condone this early simplistic and antiscientific version of the present day "politically incorrect" labeling. After failing to convince the committee that condemning words is not an effective way of fighting racism, I resigned from the editorship. However, the book was published (C11), including my name as an editor, with the amputation of the chapter on variability. I do not know if UNESCO has reversed its position on this issue.

### THE WISCONSIN CONNECTION

When we came to São Paulo, in 1958, my wife Elizabeth went to work in the Department of Physics at the same University where I was; 6 years later she had an opportunity to spend one year at the University of Wisconsin. I considered it was also time for me to go back to the States, since 10 years had elapsed since I had been with Dobzhansky at Columbia. I passed my privat docent examination and, with a Fullbright fellowship, was accepted by Dr. J.F. Crow in his Department for 1 year.

I decided to take this opportunity to learn human cytogenetics and to be able to join the efforts that A.N. Cestari and Norma C. Magnelli with some students were doing in our Department to develop the subject. The circumstances were optimal for this, since K. Patau and Eva Therman were on the staff at Wisconsin and I had the luck to work in the same room with J. M. Opitz, who was developing medical cytogenetics for diagnostic purposes.

My stay at Wisconsin was extremely rewarding. Not only did I master the current techniques for studying chromosomes, but, following my population genetical impulse, wrote an article on lethal equivalents (A26). In addition, my first article about counseling (A35), in collaboration with J.M. Opitz, J.G. Leroy, and K. Patau, was published and later reproduced in A.G. Motulsky's book on the subject.

I enjoyed thoroughly the human contacts I had with staff members, students and three Latin American friends who happened to be there: Norma C. Magnelli, an outstanding Argentinian medical geneticist, Iris Ferrari, a pioneer of this specialty in Brazil, and Anita Wajntal, from my own Department.

J.F. Crow was always kind to me. I became great friends with C. ("Charley") Cotterman, who, among other gifts, played "bossa nova" on the guitar and had a fascinating personality. J.M. Opitz was much more than a mentor to me and a wonderful companion for scientific endeavors and philosophical speculations: he diagnosed and treated with incomparable competence, dedication, and love my daughter Vanessa, who was afflicted with the "cat-eye" syndrome.

### EVOLUTION OF THE Y CHROMOSOME

I came back from Wisconsin with a problem churning in my head: why is the Y chromosome smaller than the X? As early as 1914, H.J. Muller (*J Exp Zool* 17: 325-335) developed, for the case of *Drosophila*, the idea that recessive mutations in the Y chromosome are protected against elimination by the normal alleles in the X and accumulate, producing a progressive "degeneration" of the Y. Deletions of regions thus degenerated have no adaptive value and are well afforded. As a consequence, the Y decreases.

However, Dr. Crow called my attention to an article by R.A. Fisher (*Am Natur* 69:446-455, 1935), where this theory is refuted under the assumption of panmixy. It occurred to me that, lifting this restriction, to make it more realistic, might invalidate Fisher's argument. When we included normal levels of consanguinity in his demonstration, it became compatible with the "degeneration" theory. We also calculated the accumulation of lethal genes in the Y for different levels of consanguinity occurring in human populations. This rehabilitation of Muller's theory extended to man was published in a Brazilian research journal (A34), and also included in another article in the *Brazilian Journal of Genetics* (A83).

During a short trip to the States, while I was dealing with this problem, I met Dr. M. Nei and discussed with him my refutation of Fisher's refutation. Later on he wrote me asking if I would mind him publishing a version of it using more powerful mathematics. I told him I would be delighted and he published it (*Am Natur* 104:311-322, 1970). In a recent article (W.R. Rice, *Science* 263:230-232, 1994), an experimental demonstration of the "degeneration" process is presented in *Drosophila* and it is clarified that it occurs "in finite populations", therefore with consanguinity.

I reasoned that the random accumulation of deleterious mutations and deletions in man must have led to



different degrees of shortening of the Y in different phylogenetic lines within a race and even more so in different races, and we actually found that the Y varies in size significantly more than any other human chromosome in a sample of Caucasians (A66) and that South American Indians are intermediate between Japanese and Italian men and differ significantly from both in the mean size of the Y (A83).

When the Y chromosome becomes minute, with the risk of being lost, its translocation to the X or to an autosome can "save" it. Therefore, the long-term evolution of the Y can be viewed as a sequence of cycles involving gradual loss of material separated by sudden events which tend to reconstitute its size. Phases of this cycle have been illustrated by our group of vertebrate cytogeneticists in different species (A36, 39, 53, 63).

We also studied the expression of the H-Y antigen in cases of abnormal sexual development (A74, 76, 80, 84, 92, 97).

When W. H. Price (*Lancet* i:1106-1108, 1968) showed that one third of the XYY men have an increased P-R interval in their electrocardiograms, we confirmed the reverse association, ascertaining 12 men with this peculiarity and finding that one of them had an extra Y (A45).

### CYTOGENETICS

Back from Wisconsin, in 1964, I encouraged the formation, at the University of São Paulo, of a group interested in animal evolutionary cytogenetics (A21, 36, 39, 53, 63). It was also natural, at the time, to start working in human cytogenetics, both normal (A66, 83) and medical (A24, 38, 49, 52, 67, 69, 76, 80, 84, 91, 92, 100), and to study the effect of drugs on chromosomes (A49, 51, 55, 56, 58, 74).

Besides the work on the variability of the Y chromosome, one line of research that proved especially valuable were our studies on the fragile-X syndrome, at that time yet unnamed.

Five years before the description by G. Turner et al. (*J Med Genet* 12:367-371, 1975) of sex-linked mental retardation associated with macro-orchidism, we re-

ported, in the Proceedings of the II Congress of the International Association for the Scientific Study of Mental Retardation (Warsaw, 1970, p. 745), the first nine subjects (in the same family) affected with severe sex-linked mental retardation and macrogenitalia. The discovery of this association was made by J. A. Escalante, a physician from Peru who was working with us toward his PhD. A full account of the family followed (A42). We did not study the chromosomes of these subjects at the time, but showed later that they had the fragile-X. Escalante also described in his thesis (F6) another family with three brothers with severe, and two sisters with mild mental retardation, all of them having a marker chromosome with a subterminal constriction in part of their metaphases (karyotypes studied by Angela M. Vianna-Morgante). Being unaware of the report of H.A. Lubs (*Am J Hum Genet* 21:231-244, 1969), on the marker X chromosome, we published the pedigree of this family and a metaphase with the marker chromosome, together with a brief description, in our book on medical genetics (A48), in 1973. The third publication on the subject came only 3 years later (F. Giraud et al., *Hum Genet* 34:125-135, 1976).

As we see it, the investigations of Dr. Escalante led to the first report of a family with macro-orchidism (which we called macro-genitalia) associated with X-linked mental retardation, severe in nine hemizygotes and mild in one heterozygote (A42) and to the second report on the association of fra-X with X-linked mental retardation both in males and females (A48). In this family macro-orchidism was not present, causing our failure to recognize that the two syndromes might be related. Later on, we suggested the eponym "Escalante syndrome" for the fragile-X mental retardation (A94).

### MEDICAL GENETICS AND COUNSELING

Having been through plant and Drosophilida taxonomy, *Drosophila* genetics, human population genetics, evolutionary cytogenetics and human cytogenetics I was naturally led to medical genetics.

I had graduated as a physician, but, knowing that I was not well prepared in medical matters, I never

TABLE I. Risks (%) of Manifestation of Huntington Disease for Children of Affected Persons in the Next 5, 10, and 15 Years and in the Rest of Their Lives (Total Risks) for Populations of Low Mortality Rates (From A73)

Age in years	Next 5 years	Next 10 years	Next 15 years	Total
10	—	1	2	48
15	—	2	4	47
20	1	4	9	47
25	3	7	15	47
30	5	12	21	45
35	8	17	26	43
40	9	20	29	38
45	12	21	27	32
50	10	17	21	23
55	8	12	14	14
60	5	7	7	7
65	2	3	3	3
70	1	1	1	1

worked as a physician. My zoology professor Lauro Travassos, who was also a non-practicing physician, used to advise us to obtain our M.D. degree, if we wanted to be scientists, because, in Brazil, this degree opens all doors. I never had the opportunity to test this aphorism, except by being treated by doctors without charge, until I started investigating genetic cases: my medical degree was showing its worth, when it was a matter of obtaining data and examining patients in clinics and hospitals.

Requests for karyotypes started arriving and we had to attempt a diagnosis for children with abnormalities and explain the prospects to the families and doctors. Fortunately by that time excellent reference books started to appear and I learned much consulting them with respect to each patient.

The first case in our register (February 10, 1966) was a girl with testicular feminization, demonstrated by her XY karyotype. The second had Down syndrome and the third was a case of Stein-Leventhal syndrome. Thirty years later, we have reached register number 14,000. Although receiving fewer than 500 families per year, our counseling service provided abundant material for study, because Brazilian families used to be large.

We have had several physicians working with us to specialize in medical genetics. Eight of them obtained a Master degree and five a PhD in our laboratory. We had also a number of biologists pursuing graduate work, often on the genetics of a particular disease. This is the reason we also publish continuously on aspects of medical genetics not depending on cytogenetics (A41-43, 48, 50, 54, 70, 74, 77, 79, 82, 86-90, 95-98, 100, 107, 108, 111) and specifically on data and techniques important for counseling (A35, 47, 57, 62, 64, 65, 68, 72, 73, 75, 78, 81, 93, 101, 104). I also wrote a number of reviews and book chapters (A27-33, 46, 71, 85, 105, 106, 128-130).

### COUNSELING IN HUNTINGTON DISEASE

Many persons with a parent affected with Huntington disease do not want to submit to the test which decides if they will manifest it or not. Without the test, they are told that their risk is 50%, which is wrong, unless they are newborn infants. This is because as they get older and advance along the distribution of age-of-onset without developing the disease, their probability of being heterozygous and getting sick decreases. On the other hand, the probability of manifestation in a given age interval for a heterozygote increases with age as he or she approaches the age bracket of highest risk. To solve this complex situation and set risks according to age, a distribution of onset ages and a life table for the same population are needed.

We (A62, 73) managed to get together the documents needed and develop a method to estimate the risks of having the disease, for each age bracket, in populations with three different types of lifetable: for high, medium, and low mortality. Table I gives the estimates for populations with low mortality (first world countries).

Our original papers (A62, 73) also give estimates of risks for a grandchild of an affected person, according to the age of the parent linking them.

It is a public fact that the mother of Dr. Nancy Wexler

had Huntington disease. Repeating a common mistake, Roberts (*Science*, 247:624-627, 1990) state: "Nancy Wexler herself has a 50% chance of developing Huntington's." Actually, Dr. Wexler, being now about 50 years old, has a probability of 27% of developing the disease any time for the rest of her life (Table I).

In general, people having a parent affected think that their risk is 50% at any age. Many of them refuse the test and live in fear. Explaining to them the true risk, which is always smaller than this, the counselor decreases their anguish and establishes a new basis for them to reconsider their decision about the test.

The total risks in the last column in Table I can now be checked or corrected comparing them to the empirical frequencies of positive tests, tabulated according to age, from the records of laboratories performing them.

### THE OAS MULTINATIONAL GENETICS PROGRAM

Since some countries in Latin America already had good research centers in genetics, OAS decided, in 1968, to send to three of them graduate students from other countries in Spanish America, thus helping in the training of geneticists which was until then carried out locally or in the States and Europe. Our laboratory took charge of those interested in human genetics while a center in Chile received prospective animal geneticists and another in Argentina gave the training in plant genetics.

In this successful program, unfortunately discontinued 5 years later, four Peruvians, three Bolivians, and one Colombian obtained with us a Master degree and two of them also a PhD, becoming leading geneticists in their countries.

The OAS Program produced also indirect benefits sponsoring visiting professors: we had with us Eduardo Castilla, Rafael Elejalde, George Frazer, David Klein, Osvaldo Mutchinik, Norma C. Magnelli, Glaucia Peres Mosquera, Robert Murphy, John M. Opitz, and Francisco Saez. Our three centers also had joint seminars, where the graduate students discussed their projects, which gave me the opportunity of meeting geneticists from different Latin American countries. Also I gave courses twice in Lima, Peru. One result of these contacts was the foundation of the Associação Latinoamericana de Genética, in 1969, of which I was the first president.

### RETIRING

The laboratory of human genetics I had started on arrival to São Paulo has grown and matured considerably. It split naturally into roughly three groups: P.A. Otto coordinates population genetics and counseling, Mayana Zatz, muscular dystrophy, and Angela M. Vianna-Morgante, cytogenetics. Our group of hemophilia has been discontinued, as I failed in obtaining a contract in the University for its coordinator, Ruth Levisky. A fourth laboratory, led by Yatiyo Yonenaga-Yassuda, originated by gemmiparity from our inaugural group and takes care of evolutionary cytogenetics of rodent and other vertebrates. Denise Peccinini-Seale works with lizards. A group, of immunological genetics, led by C.A. Moreira Filho, was transferred to another Institute in

our University. Priscila G. Otto deals with human chromosomes, and is very involved with the production of textbooks on genetics. A laboratory of human evolution, led by W. A. Neves, resulted from the most recent pregnancy in our nest. Our group has also gone through a number of sporulations, responsible for many of our PhD germinating in other laboratories and cities in Brazil and abroad. As good geneticists must do, we have proliferated.

### AN INCURSION ON PSYCHIATRIC GENETICS

When I was about to retire, I became interested in behavior genetics (A110, 115, 125) and then in the genetics of mental diseases. I started counseling families with mental cases and this obliged me to learn and apply DSM-III criteria for diagnoses. I was appalled by the frequency of *propositi* having histories compatible with bipolar disorder with psychotic signs, who were diagnosed by their doctors as schizophrenics and treated with neuroleptics. This led me to write a paper (A112) showing that, given the literature prevalences of these disorders in the population, a patient with psychotic signs (of whom nothing more is known) more probably has mood disorder than schizophrenia, and that, taking into account modern estimates of empirical recurrence risks, this is true even if he or she has a first degree relative also with psychotic signs. It was a theoretical paper with an important practical conclusion: many people with psychotic mood disorder are not getting the best treatment (with lithium) because of misdiagnosis.

### Commutative Polygenes

To gain experience, I decided to get involved in field projects and carried out a pilot study to check the possible genetic relationship between alcoholism and mood disorder, suggested by some authors. I interviewed, with students, a number of alcoholics recuperating during in-patient treatment, inquiring about their relatives and setting up their pedigrees with respect to mental disorders (A102). Their families contained suspects for mood disorder significantly more often than in a control sample of families of university students. This supports the notion that alcoholism and mood disorders are somehow genetically related and that the presence of alcoholism cases in a family can be taken as a marker of genetic predisposition for mood disorder, and vice-versa.

Since both diseases are most probably multifactorial polygenic in determination (A116, 117), I developed the concept of "commutative polygenes" to explain the genetic connection between them (A114, 116, 120–123). These are polygenes capable of influencing more than one trait, depending on the joint effect of the remaining polygenes and environment factors.

The need for this concept comes from the following reasoning. If two independent multifactorial polygenic systems are responsible for the two diseases, a family showing concentration of both is explained by the chance occurrence of both systems in it; however, the incidence of mood disorder in families of alcoholics and in control families should not differ. If they do, it is because elements belonging to the alcoholism polygenic

system are also inducing mood disorder, or vice-versa. I call such elements commutative polygenes.

A pleiotropic polygene produces two effects in the same subject; while a commutative polygene reinforces a trend in one patient and another trend in a second patient, according to the influence exerted by the remaining polygenes. For an illustration, let us consider a commutative polygene *c* pushing a normal person toward extroversion. If the person happens also to have the polygenic system favoring mood disorder, *c* will help induce this disease (or mania, rather than depression); and if it is the alcoholism polygenic system which is present, *c* will push toward alcoholism (and perhaps specially toward Cloninger's type 2 alcoholism).

The concept of commutative polygenes helps to explain the clusters of diseases which appear in excess in the families with a main disorder: to the spectrum of schizophrenia belong the schizotypic and the paranoid personality disorders, atypical psychosis, schizoaffective disorder, depressive type, and mood disorder with incongruent delusions. Another spectrum gathers around mood disorders.

### Predisposition to Alcoholism

Our data (unpublished) showed, also, that middle class alcoholics (those paying for their treatment in private hospitals) had families with cases of probable mood disorder more often than poorer alcoholics, cared for in free hospitals (difference significant at the 0.01 level). Taking mood disorders as a genetic marker for alcoholism, this seems to mean that greater genetic predisposition is needed to induce alcoholism in a person submitted to middle class criticism to addiction than in one belonging to the more lenient lower economic class.

Based on this interpretation, I reasoned that the genetic predisposition to alcoholism present in female alcoholics should be greater than that in male alcoholics, because social pressure against alcoholism is obviously greater for females. To check this, we interviewed female alcoholics and found that they were more frequently from families with cases of mood disorder than the male alcoholics (difference significant at the 0.05 level). This suggests that the greater the environmental pressure against alcoholism, the greater the presence of cases of mood disorders in the families of alcoholics.

### Mood Disorder and Suicide

In another project, we studied the pedigrees of people who attempted or committed suicide and found that almost all of them had members suspected of having mood disorders (A118, 119). This was recently confirmed by one of my graduate students who compared her series with a control series and found a significant difference between them. Our conclusion is that doctors attending cases of attempted suicide must routinely survey the patient's family, looking for cases of mood disorders to be treated, as an effective way of avoiding additional suicide attempts.

### Non Confirmation of Gene Localizations

The core of the ideas in the literature trying to explain the lack of confirmation for the localizations of

genes for bipolar disorder and schizophrenia amounts to three:

- a) in spite of the power of the method used, the results were false positives;
- b) the results involved some technical or computational error;
- c) the localizations were correct but different genes can determine the disease in different families (monogenic heterogeneity).

In 1989, I submitted an alternative hypothesis (A114–116, 120–124), based on the following points:

1. Segregation analysis makes it probable that bipolar disorder has a multifactorial polygenic determination. The same model or the mixed model are adequate for schizophrenia.

2. Families suitable for gene localization must have the greatest possible number of affected members. To produce such high average genetic liability, most loci belonging to the polygenic system in these families must be homozygous for the alleles favoring the disorder and, therefore, are uninformative.

3. If only one of the polygenic loci is segregating in the family, the linkage information derives entirely from it. Such a polygene mimics a monogene, by representing the final drop of water which makes liability cross the threshold of manifestation in individuals already very near to it. This allows the localization of the polygene with an appropriate probe, as it were a monogene.

4. Even if a few loci are segregating, the distribution of the genotypes can be such as to allow the localization of one of them as if it were a monogene.

5. New families, analyzed with the same probe, most probably will give negative results, because the polygene localized before has now a great probability of being homozygous for the allele favoring the disease and thus being uninformative (item 2).

6. If the assay is repeated with the same probe, again and again, in big families (but not in groups of families), another positive result will eventually emerge, but it will remain also unconfirmed in further families because the same locus has a great chance of not being segregating in them. The habit of pooling several families in one test tends to give negative results, because the polygenic loci segregating are probably not the same in the different families.

7. The number of polygenic loci interfering with the manifestation of a disease is proportional to the average number of families required to be studied for a confirmation to occur.

8. In the meanwhile, localizations with other probes for the same disease will occur and remain unconfirmed in further families for a while. They correspond to the discovery of other polygenes influencing the liability to the same disease.

### Polymemes

R. Dawkins introduced the concept of meme, a cultural unit socially transmitted, as a counterpart to the gene. We found that it is useful to integrate this idea into the multifactorial model (A116, 121–123). Some of the environmental factors contributing with polygenes to a normal trait or a disease are culturally transmitted

and carry their weight through the generations: they are polymemes. The habit to forbid kids to take alcoholic beverages, prevalent among Irish Americans, and the opposite habit of letting them participate in the family libations, more common among the Italian Americans, can be taken as cultural alleles from a polymeme. They influence the liability to alcoholism, together with other polymemes and some polygenes. The allele “you shall not drink,” explains the lower incidence of alcoholism among Mormons as compared to popular music performers, taken by the allele “alcohol is needed for inspiration,” from the same meme.

A molecular genetic treatment for alcoholism is not known, but an effective polymeme allele has been engineered, which is very effective in lowering the liability to alcoholism: Alcoholics Anonymous.

An important shift in “population polymemetics” was precipitated by the warnings from the Surgeon General to the effect that “smoking is dangerous to your health”. The population frequency of this memic allele, which influences the multifactorial system controlling lung cancer and other diseases, has taken space from opposite alleles, which, however, were reinforced more recently by new memes promoted by the tobacco industry, such as the Joe Camel cartoon. They show that smoking induces mutations, also at the memetic realm.

We can even talk about a commutative polymeme: for instance, a meme of profound sense of responsibility, culturally inherited through education, can push us toward organization, obsession, depression, or alcoholism, according to the nature and strength of the other polymemes and polygenes with which it is interacting.

### BACK TO THE BEGINNING

Although approaching my eightieth birthday, I keep reasonably busy. I collaborate with colleagues in research (A126, 127, 131, 132), deliver invited lectures and participate in round tables and seminars. I also maintain my old habit of witting on science teaching (E1–42) and a little on scientific policies and the history of science (B1–17 and the very biased piece I am writing right now).

Along with this, I am concentrating now on something that I started doing 35 years ago: writing texts for the youth. With Priscila G. Otto and Paulo A. Otto, I am making a revised edition, in one volume, of our two books on human and medical genetics (D8, D9). I am also starting an up-to-date version of the *Biologia na Escola Secundária*. If an individual life follows a cycle, as some people believe, it is nice to make it round.

I do not feel signs of physical or mental impairment, except for a stationary atherosclerosis and slight decrease in my vision, audition, and memory, which do not interfere significantly with my endeavors. I appreciate more deeply than ever all the gifts and enjoyments that life insists in bringing to me everyday, especially through my relatives, friends, colleagues, and students. I take great satisfaction from being in line with the replicators which constructed, up to now, my four children, eight grandchildren, and six great-grandchildren, a clear proof that I am a link in the uninterrupted chain of genes and their vehicles started four

billions years ago and stretching to the unforeseen future. But I relish even more to be immersed in the majestic stream of science, which lets me know who I am, from where I came and what final fate faces me.

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## F: Orientation of Master and PhD Theses

## Master Theses

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- F2. Yatiyo Yonenaga. “Estudos cariotípicos de espécies de morcegos (Chiroptera)” (15.07.68).
- F3. Denise M. Peccinini Seale. “Cariótipos e mecanismo de determinação do sexo em algumas espécies de lacertílios brasileiros (Iguanidae e Teiidae)” (26.07.69).

- F4. Ricardo Jaime Descailleaux Dulanto. "Frequência de cromatina sexual em diferentes regiões do corpo feminino" (22.10.70).
- F5. Nomaiaci Ramos Ferreira. "Tratamento colchicínico e células aneuplóides" (02.11.70).
- F6. Júlio Aníbal Escalante Forton. "Malformações congênicas de membros e deficiência mental" (19.11.70).
- F7. Vilna de Vasconcelos Maia. "Investigações de associações entre dermatóglifos e variáveis normais (grupos sanguíneos ABO) e patológicas (mongolismo)" (19.11.70).
- F8. Mayana Zatz. "Detecção de heterozigotas quanto ao gene da distrofia muscular progressiva tipo Duchenne" (10.12.70).
- F9. Angela Maria Vianna-Morgante. "Translocações cromossômicas e mongolismo" (17.12.70).
- F10. Luz Becker Rodrigues. "Investigação de anomalias cromossômicas em homens criminosos de alta estatura" (23.06.71).
- F11. Ruth Blay Levisky. "Estudo genético das distrofias musculares progressivas e aconselhamento genético" (28.06.71).
- F12. Cleide Largman Borovik. "Variabilidade dos cromossomos no cariótipohumano" (21.07.72).
- F13. Carlos Rogério Mello da Silva. "Estudo clínico de 123 casos de mongolismo" (05.12.72).
- F14. Nicole Stephanie Loghin. "Avaliação dos critérios de identificação do cromossomo Y humano" (05.02.73).
- F15. Iêda Maria Orioli Parreiras. "Estudo da sensibilidade aos andrógenos em dois casos de pseudo-hermafroditismo familiar" (16.02.73).
- F16. Jorge Rolando Olivares Plaza. "Estudo prospectivo sobre malformações congênicas em recém-nascidos" (26.06.73).
- F17. Evani M. Viegas Péquignot. "Estudo de mosaicismo cromossômico em genitores de pacientes com síndrome de Down (mongolismo)" (21.03.74).
- F18. Sanae Kasahara. "Estudo de mosaicismo cromossômico em genitores de pacientes com mongolismo (síndrome de Down)" (21.03.74).
- F19. Carmen R.S. de Taboada. "Estudo e Aconselhamento Genético em 68 casos de cegueira" (29.05.74).
- F20. Juan Manuel Gonzalo Taboada-Lopez. "Aspectos genéticos do glaucoma infantil primário" (21.06.74).
- F21. Maria Victoria Monsalve. "Diferença do comprimento do cromossomo Y entre italianos e japoneses" (23.09.74).
- F22. Ana Maria G. Campos. "Alterações citogenéticas nas células de medula óssea de hamsters expostos a radiação de corpo inteiro pelo cobalto-60" (23.09.74).
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- F24. Decio Brunoni. "Estudo de cinco sinais genético-clínicos em uma amostra de recém-nascidos" (15.04.77).
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- F30. Silvia Luzia Frateschi Trivellato. "O ensino de Genética em uma escola de 2º grau" (17.12.87).
- F31. Patrícia de Campos Pieri. "Conhecimentos e crenças em amostra de 348 gestantes que se dirigem ao diagnóstico pré-natal em São Paulo: Vol. I e II" (11.04.91).
- F32. Sandra Odebrecht Vargas Nunes. "História familiar em tentativas de suicídio" (13.10.93).
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- F33. Júlio Aníbal Escalante Forton. "Estudo genético da deficiência mental" (28.07.71).
- F34. Denise M. Peccinini Seale. "Variação cromossômica em populações partenogenéticas e bissexuadas de *Cnemidophorus lemniscatus* (Sauria, Teiidae) no Vale do Amazonas" (12.02.73).
- F35. Yatiyo Yonenaga. "Polimorfismos cromossômicos em roedores brasileiros" (13.02.73).
- F36. Nilda Martello. "Riscos de manifestação da Coréia de Huntington em diferentes idades e aconselhamento genético" (29.03.73).
- F37. Ruth Blay Levisky. "Estudo genético e Aconselhamento Genético em miopatias hereditárias" (11.10.73).
- F38. Mayana Zatz. "Atividade da creatino-fosfoquinase e estudos de ligação em Distrofias Musculares Progressivas de herança ligada ao X" (27.05.74).
- F39. Angela Maria Vianna-Morgante. "Anomalias cromossômicas estruturais no homem: Estudo citogenético de seis famílias" (08.08.74).
- F40. Cleide Largman Borovik. "O cromossomo Y grande em homens selecionados por estatura, comportamento anti-social e anomalias eletrocardiográficas" (14.04.77).
- F41. Paulo Alberto Otto. "Estudo matemático de alguns modelos de cruzamentos preferenciais" (02.08.77).
- F42. Maria Victoria Monsalve. "Tamanho do cromossomo Y em japoneses, índios e italianos" (27.06.78).
- F43. Carlos Alberto Moreira Filho. "O antígeno H-Y e a genética da determinação primária do sexo" (04.06.80).
- F44. Thomaz Rafael Gollop. "Estudo genético-clínico das disostoses mandibulofacial e frontofacionasal" (11.12.81).
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- F46. Marta Pinheiro. "Displasias ectodérmicas do grupo A: classificação, etiologia genética e descrição de duas afecções novas" (12.12.83).
- F47. Tamara June Lister. "Albinismos: diagnóstico diferencial e genética (19 casos estudados)" (27.04.84).